Amendment to the Specification:

ABSTRACT

Please replace the Abstract with the following amended Abstract:

A method for identifying compounds which selectively modulate expression of polypeptides which are important in the growth and survival of Candida albicans is disclosed. Also disclosed are sequences that are important for the survival and growth of Candida albicans and compounds identified in accordance with the method.

DISCLOSURE

Page 18, lines 10-11, please amend the paragraph as follows:

Identification of novel drug targets in C. albicans by anti-sense and disruptive integration.

Page 37, lines 7-24, please amend the paragraph as follows:

To verify that the growth-effect was due to the interference with the identified gene and to support the specificity of the antisense effect, single allele knock-outs were made in 6 identified genes using the URA-blaster method (Fonzi and ~~irwin~~ Irwin, 1993). Disruption of one allele of a gene should in theory lead to ~ 50 % reduction in gene transcript. In practice however we have observed reductions varying between 10 and 100 % of normal level. This can probably be explained by the fact that ~~not always~~ both copies of a gene are not always functional. That only a single integration at the ~~corerct~~ correct site had occurred for each of the disruption cassettes was verified by PCR and Southern blot analysis. Growth curves were measured; three disruptants showed impaired growth, suggesting that a gene required for growth or survival was targeted. Experiments to take over control of the second allele of each gene -by promoter replacement- are ongoing.

Page 30, lines 7-17, please amend the paragraph as follows:

The method proposed is based on observations (Sandbaken et al., 1990; ~~Hinnebusch and Liebman 1991~~ Hinnebusch and Liebman, 1991, Protein synthesis and translational control in Saccharomyces cerevisiae, pp. 627–735 in The Molecular and Cellular Biology of the Yeast

Saccharomyces: Genome Dynamics, Protein Synthesis, and Energetics, Vol. I, edited by J. R. Broach, J. R. Pringle and E. W. Jones. Cold Spring Harbor Laboratory Press, Cold Spring Harbor, NY; Ribogene PCT WO 95/11969, 1995) suggesting that underexpression or overexpression of any component of a process (e.g. translation) could lead to altered sensitivity to an inhibitor of a relevant step in that process. Such an inhibitor should be more potent against a cell limited by a deficiency in the macromolecule catalysing that step and/or less potent against a cell containing an excess of that macromolecule, as compared to the wild type (WT) cell.

Page 38, lines 3-20, please amend the paragraph as follows:

In our genomic screen, integration of the library plasmid can happen either at the endogenous GAL1 promoter locus or, more frequently, at the locus corresponding to the plasmid insert. The latter results in a gene duplication with the first copy of the gene flanked by two convergently oriented promoters. The use of such a "collision construct" has previously been described in screening for inhibitors of transcriptional activation in mammalian cells (patent WO 97/10360; Giese K.). If RNA polymerase II complexes start from both the upstream and downstream, oppositely oriented, promoter regions, they may collide thereby preventing the formation of a full-length mRNA transcript. The second copy of the gene ~~has no more~~ no longer has a promoter and is probably 5' crippled as the original inserts cloned into the library have an average length of ~1.5 kb while ORFs in C. albicans have an average length of ~~...~~ 600 bp and we ourselves identified ORFs of (unknown) genes larger than 7 kb.